Trunk control in low back pain

Willigenburg, N.W.

2012

Chapter 4

Precision control of an upright trunk posture in low back pain patients

Nienke W. Willigenburg

Idsart Kingma

Jaap H. van Dieën

Clinical Biomechanics 2012, doi: 10.1016/j.clinbiomech.2012.06.002

Abstract

Low back pain appears to be associated with impaired trunk postural control, which could be caused by proprioceptive deficits. We assessed control of trunk posture in conditions requiring high and low precision, with and without disturbance of proprioception by lumbar muscle vibration. Twenty a-specific low back pain patients and 13 healthy controls maintained a self-chosen upright trunk posture. Initial frontal and sagittal plane angles of an opto-electronic marker on the 12th thoracic spinous process defined the center of a target area on a monitor. Subjects were instructed to stay within that target and visual feedback was provided when they left the target. The precision demand was manipulated by changing target size. The standard deviation of trunk angle quantified precision and mean Euclidian distance to target center quantified accuracy. Ratios of antagonistic co-activation were calculated from trunk muscle electromyography recordings. With the small target, visual feedback was present intermittently and patients controlled their trunk as accurately and precisely as healthy controls. For the large target, subjects mostly stayed within the target, and patients were on average 0.18^{o} (31%) less accurate than healthy controls ($p=0.025$), due to a larger postural drift. Lumbar muscle vibration deteriorated control over trunk posture in both groups and ratios of antagonistic co-activation did not differ between groups or conditions. These results indicate that the weighting of proprioceptive feedback from lumbar muscle spindles did not differ between groups and that low back pain patients were less able to detect low frequency drift in posture.

Introduction

Differences in motor behavior between low back pain (LBP) patients and healthy control subjects have been demonstrated in a variety of tasks, e.g. during walking and in response to several perturbations [29-30, 96-98]. Such differences may be causal for LBP, but could also be effects of LBP. Changes as a result of LBP might subsequently either promote or protect against recurrence and/or chronicity of LBP, implying that differences in motor behavior are not necessarily appropriate targets for intervention [33]. Nevertheless, motor behavior has been targeted in conservative interventions to LBP, and with some success but rather limited effect sizes [15].

Published evidence indicates that LBP patients may have impaired control over trunk posture and movement [96, 99-100] and if present such impairments could promote recurrence and/or chronicity of LBP. Impairments of trunk control are reflected in reduced accuracy and precision of trunk movement. Accuracy is high when the mean difference between a desired movement/posture and the actual trunk movement/posture is small, whereas precision is high when the variability of trunk movement is low. Descarreaux and colleagues studied repositioning accuracy in moving toward different trunk postures. No difference in accuracy was found between LBP patients and healthy control subjects, but a subgroup of patients needed substantially more practice trials to achieve similar levels of accuracy and moved slower during the repositioning tasks [99]. Willigenburg and colleagues studied accuracy in a tracking task that required circular movements of the trunk. On average, LBP patients made larger errors than healthy subjects [100].

Possibly, trunk control impairments in LBP patients are caused by impaired trunk proprioception. Previous studies with paraspinal muscle vibration, which is known to perturb proprioceptive feedback from muscle spindles [101], showed that the weighting of proprioceptive feedback from lumbar muscle spindles relative to proprioceptive feedback from calf muscles is lower in LBP patients than in healthy control subjects [52, 54]. In addition, when performing a tracking task with the trunk, accuracy was less affected by lumbar muscle vibration in LBP patients than in healthy control subjects [100]. These findings may point to disturbed proprioceptive information from the trunk muscles, reduced reliance on such information, or both.

If indeed proprioception is impaired, this might be compensated for by trunk stiffening through antagonistic co-activation [42]. Indications for the use of such a stiffening control strategy have indeed been observed in LBP patients performing slow sagittal plane trunk movements [37], but not in the trunk tracking task mentioned above [100]. This may suggest that, whereas LBP patients show increased trunk muscle co-activation in general, and do not further increase co-activation under precision demands, healthy subjects increase co-activation to the level of LBP patients under precision demands. In line with this, we found no modulation of co-activation in healthy subjects over tasks with varying, but all quite high precision demands [91].

In the present study, we evaluated accuracy and precision of the control of an upright trunk posture, as well as trunk muscle co-activation levels in LBP patients and healthy controls, in conditions with high and low precision demands. To evaluate the effect of proprioception disturbance, experimental tasks were performed in conditions with and without lumbar muscle vibration. We hypothesized that LBP patients would be less accurate and less precise in conditions without lumbar muscle vibration. Furthermore, because LBP patients may either have impaired trunk proprioception or may rely less on trunk proprioception than healthy subjects (as described above), we hypothesized that disturbance of proprioception by lumbar muscle vibration would have a larger effect in healthy subjects than in LBP patients. Finally, we hypothesized that trunk muscle co-activation would be higher in LBP patients in the non-precise condition only.

Methods

Subjects

Inclusion criteria for the LBP patient group included self-reported LBP for at least the past six weeks, specific diagnosis excluded by a general practitioner or physical therapist, no previous surgery on the spine, no other conditions (e.g. neurological or mental disorders, allergy to plaster) hindering participation or performance, age between 18 and 65, and a score ≤105 on a yellow flags screening questionnaire [94] which indicates low to moderate level of risk for chronicity based on psychosocial factors.

Twenty LBP patients (11 male, 9 female) who fulfilled these criteria and 13 healthy controls (9 male, 4 female) with no history of LBP participated in the experiment. No

significant differences (all p≥0.455) between subject groups existed in age and BMI (averages (SD) of 33.4 (15.5) vs. 34.3 (11.9) years and 23.6 (3.0) vs. 22.9 (2.4) kg/m^2 for LBP patients and healthy controls, respectively). LBP patients scored 2.7 (1.9) cm on a 10 cm visual analogue pain scale at the start of the measurements. The experimental protocol was approved by the local medical ethics committee and all subjects provided written informed consent before participating.

Experimental setup

Figure 4.1 shows the experimental setup in which subjects maintained a semi-seated position with their pelvis fixed. To minimize upper limb contributions to trunk postural control, both hands were placed on top of the head. Opto-electronic markers were placed on the spinous process of T12 and at pelvis height on the frame (fixed point in space). Trunk angles in the frontal and sagittal planes of motion were defined as the angles of the line through these two markers with respect to the vertical.

A monitor in front of the subjects provided real-time visual feedback of trunk angle (delay max. 10 ms), with a spatial resolution of 0.05^o per pixel (600x600 pixels with a range of 30^o in both directions). Trunk angle changes in the frontal plane (lateral flexion) corresponded to movements of a cursor along the X-axis (left-right), while trunk angle changes in the sagittal plane (flexion and extension) corresponded to cursor movements along the Y-axis (up-down).

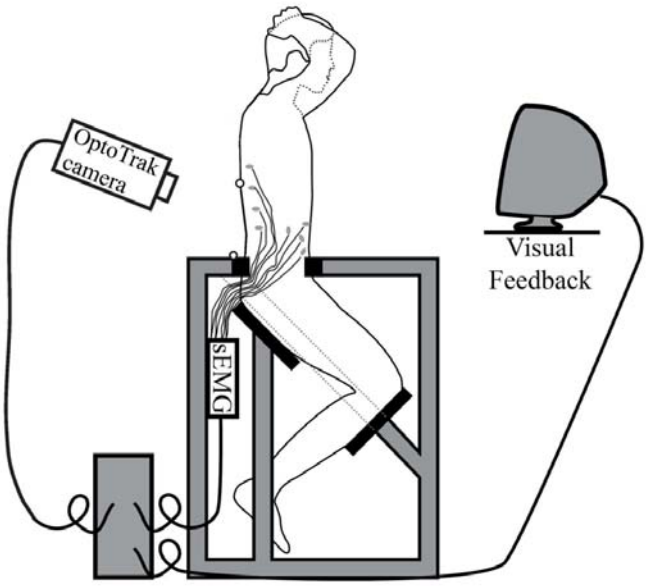

Figure 4.1. Experimental setup, by Paul van Drunen.

At the beginning of each trial, the subject was instructed to adopt a neutral posture. Then data collection started and the first data sample was used to determine the neutral trunk angle, which was projected at the center of the screen, surrounded by a yellow square target area. Subjects were instructed to stay within the target area for 30 seconds during each trial. In the first 5 seconds, a black dot (diameter 92 pixels), representing the trunk angle in real-time was visible. After that, visual feedback was only provided when subjects left the target. When that happened, the side at which the subject crossed the target border turned red, indicating that the trunk angle should be adjusted in the opposite direction.

To manipulate the precision demand, the target square was small in half of the trials and large in the other half. The small target (100x100 pixels) imposed a high precision demand; the margin in trunk angle was only 0.2^{o} (4 pixels) from the target center in each direction. The large target (200x200 pixels reflecting a margin of 2.7^{o} (54 pixels)) was chosen such that subjects could stay within the target without much effort, to impose no or only a very low precision demand.

Electromyography (Porti 17, TMS, Enschede, The Netherlands, 22-bits AD conversion after 20x amplification, input impedance $>10^{12}$ Ω, CMRR >90 dB) was recorded of four abdominal and four back muscles, both left and right, using pairs of surface electrodes (Ag/AgCl, inter-electrode distance 25 mm) that were attached to the skin after shaving and cleaning with alcohol. Electrodes to record thoracic back muscle activation were placed 4 cm lateral to T9 (thoracic part of m. longissimus) and 6 cm lateral to T11 (thoracic part of m. iliocostalis) spinous processes. Activation of lumbar back muscles was recorded 6 cm lateral to the L2 (lumbar part of m. iliocostalis) spinous process and 3 cm lateral to the midpoint between the spinous processes of L3 and L4 (lumbar part of m. longissimus). At the ventral site, electrodes were placed 3 cm lateral to the umbilicus or somewhat lower when a tendinous intersection was present there (m. rectus abdominus), 3 cm medial to the anterior superior iliac spine (ASIS) (m. obliquus internus), in the mid-axillary line between the iliac crest and the 10^{th} rib (lateral part of m. obliquus externus) and at the crossing point of a horizontal line through the umbilicus and a vertical line through the ASIS (anterior part of m. obliquus externus). Muscle activation was recorded at a sample rate of 1000 samples/s and a pulse signal was used to synchronize the kinematics and EMG data.

To disturb proprioceptive feedback from lumbar muscle spindles, vibration was applied to the paraspinal muscles bilaterally (in between L3 and L4) in half of the trials. The

order of trials with and without lumbar muscle vibration was counterbalanced between subjects. In trials with lumbar muscle vibration, a motor (Maxon Graphite Brushes S2326.946 driven by a 4-Q-DC Servo Control LSC 30/2 in a velocity loop) rotating an eccentric mass at a frequency of 90 Hz was attached to the subject's lower back with neoprene elastic bands (Figure 4.2). The motor was turned on after defining the neutral posture, but within the first 5 seconds of the trial. Two repetitions of each condition were performed, resulting in eight trials (2 targets x 2 vibration conditions x 2 repetitions) per subject.

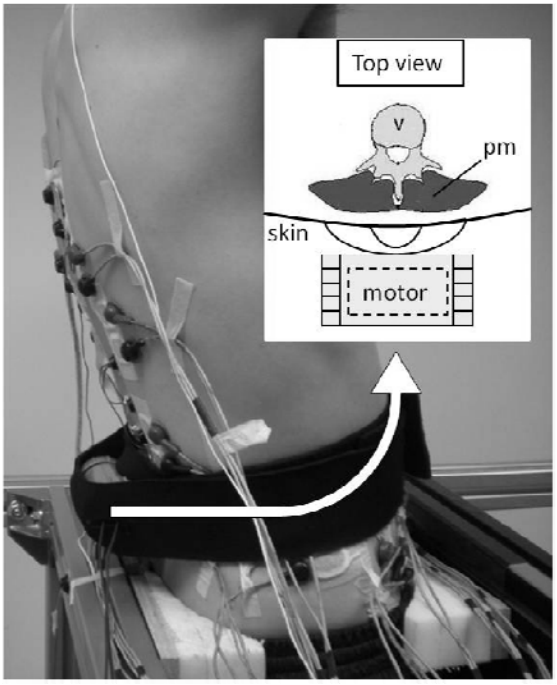

Figure 4.2. Vibration device as attached over the paraspinal muscles (pm) at the level of L3/4. The vibrating motor was stored in a plastic cylinder, and a U-shaped piece of solid plastic between the motor and the skin ensured bilateral muscle vibration while leaving the spinous process of the lumbar vertebra (v) free.

Data analysis

Due to practical issues such as turning on the vibrating motor and transitioning from continuous to intermittent visual feedback, the first 5 seconds of each trial were discarded from further analysis. Opto-electronic data were 2.5 Hz low pass filtered (2^{nd} order bi-directional Butterworth) before trunk angles in the frontal and sagittal planes were calculated (see above), since higher movement frequencies are not likely to occur given the trunk's high inertia. To illustrate the outcome measures of interest, Figure 4.3 shows a typical example of trunk angle variability in the frontal and sagittal planes of motion. The percentage time on target was calculated as a measure of task performance. To quantify

precision, the standard deviations (SDs) of the trunk angle in both planes of motion were calculated and then averaged. To quantify accuracy, trunk angles in both planes were averaged over time and the Euclidian distance to the target center (the length of the hypothenuse from frontal and sagittal plane mean angles with respect to the neutral angle) was calculated in degrees.

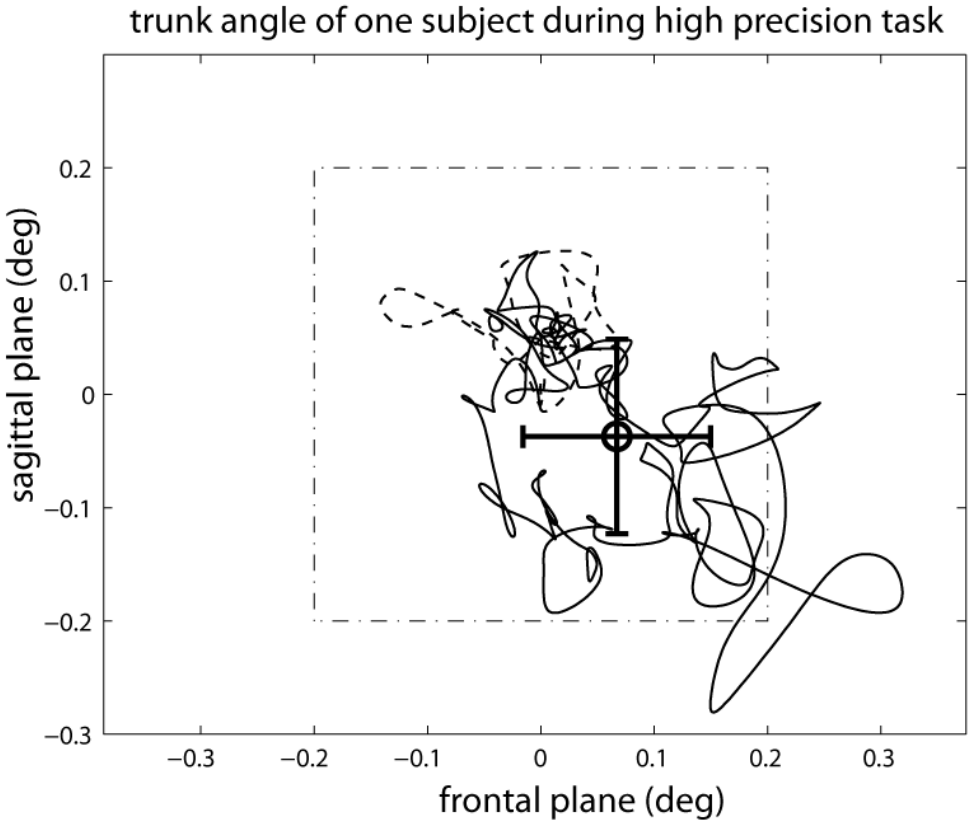

Figure 4.3. Typical example of one subject performing a trial with a small target without vibration. Target boundaries are dash-dotted and the target center (neutral posture) is marked with an asterisk. The first 5 seconds (dashed) were discarded from analysis. Mean and SD over the consecutive 25 seconds used for analysis (solid) are shown as a circle with error bars. Note that the Euclidian distance from asterisk to circle represents accuracy and that precision was quantified as the average over SDs in both planes.

EMG signals were 250 Hz high-pass filtered to remove low frequency contamination (e.g. the electrocardiogram and hum) and to improve estimates of muscle activation [102-103]. Subsequently, absolute Hilbert amplitudes were calculated and ratios of co-activation were obtained by dividing EMG amplitude averaged over the abdominal muscles by EMG

amplitude averaged over the back muscles. The lumbar m. longissimus signals were contaminated by the muscle vibration, therefore they were discarded from analysis.

Statistics

For accuracy, precision and co-activation ratios, repeated measures ANOVAs, with group (LBP patients vs. healthy controls) as between-subject factor and target size (large vs. small) and vibration condition (with vs. without) as within-subject factors, were performed in SPSS 16.0. When a significant interaction with the factor group was found, follow up ANOVAs were performed to test the effect of group for the within-subjects factor separately. Percentage time on target was almost always 100% for the large target. Therefore, for percentage time on target, ANOVA's were only applied to the small target and the factor target was omitted. The level of significance was set at $\alpha<0.05$ and p-values between 0.05 and 0.10 were marked as non-significant tendencies.

Results

As noted above, when the target was large, percentage time on target was almost always 100% in both groups and vibration conditions. When the target was small, no significant main effect of group (82.5% and 78.9% for the healthy controls and LBP patients, respectively; $p=0.179$) or group x vibration interaction ($p=0.737$) was found. However, time on target was significantly affected by vibration condition (86.5% without versus 75.0% with lumbar muscle vibration; $p<0.001$).

For both groups, the effects of target size and vibration condition on precision and accuracy of trunk control are shown in Figure 4.4. For precision, no main effect of group ($p=0.507$) or interaction with group (all $p\geq0.424$) was found. Significant main effects of target size and vibration (both $p\leq0.003$) indicated that both LBP patients and healthy subjects were more precise when the target was small and that precision decreased with lumbar muscle vibration in both groups. A significant interaction between target and vibration condition ($p=0.040$) indicated that effects of vibration on precision were smaller when the target was small. Apparently, the visual feedback when subjects left the target compensated for the disturbance of proprioception.

For accuracy, a significant main effect of group ($p=0.016$) as well as a significant group x target interaction ($p=0.044$) was found. This indicated that LBP patients deviated

significantly further from the target center than healthy controls and that this effect was larger with the large target. Follow up ANOVAs for the different targets separately revealed no difference between groups (p=0.164) when the target was small, while the average deviation from the target center in LBP patients was 0.18^{o} (31%) larger than in healthy control subjects when the target was large (p=0.025). In addition, effects of target size, vibration condition and their interaction were significant (all p<0.001). Effects of lumbar muscle vibration were clearly visible in the sagittal plane, where both groups demonstrated an offset towards extension, mainly in conditions with a large target (Figure 4.4).

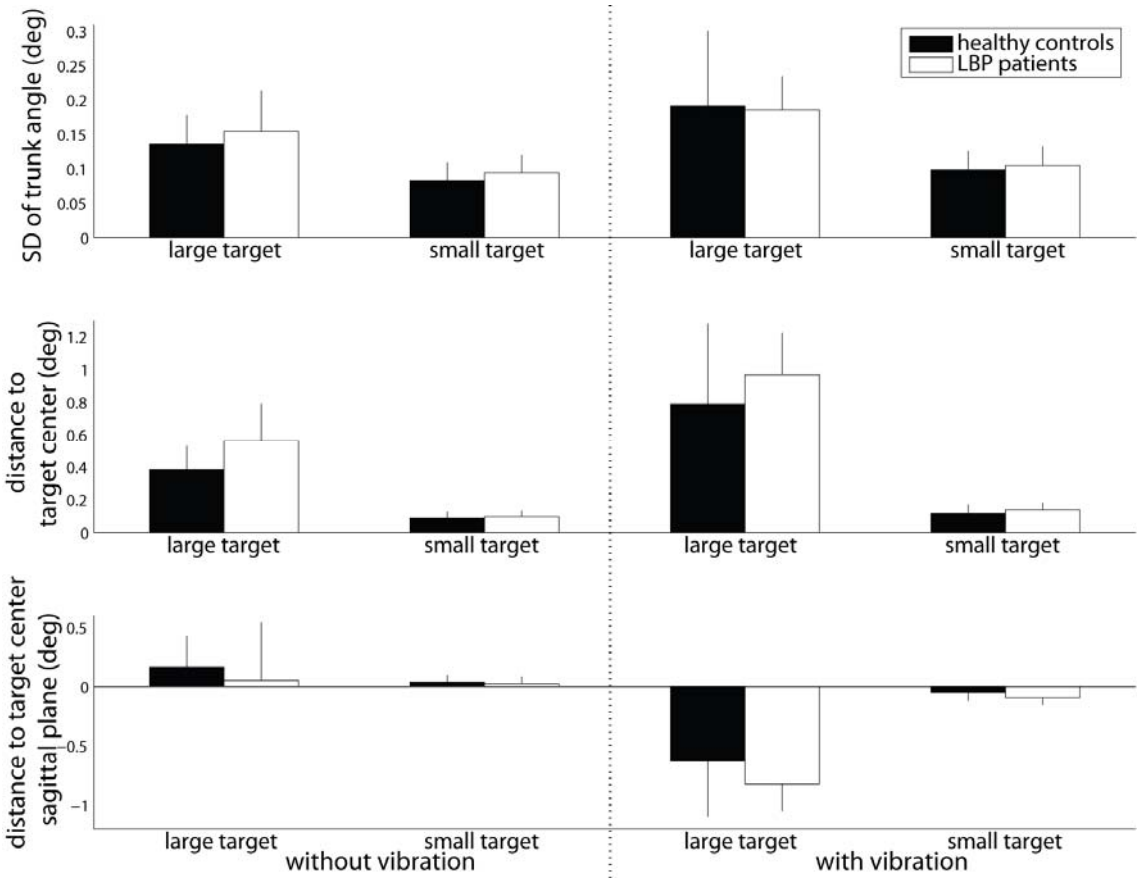

Figure 4.4. Precision and accuracy of trunk control for both targets and groups in conditions without (left panel) and with (right panel) lumbar muscle vibration. SD of trunk angle (upper panel) and Eucledian distance to target center (second panel) were calculated over sagittal plane and frontal plane trunk angles combined. Note that a large SD reflects low precision and, similarly, that a large distance to the target center reflects low accuracy. The lower panel illustrates the effect of lumbar muscle vibration on accuracy in the sagittal plane. Error bars represent SDs over subjects within groups.

To gain further insight of the difference between LBP patients and healthy controls in accuracy that did not coincide with an effect of group on precision, we evaluated how the Euclidian distance to the target center changed over time. By comparing the average over the last 3 seconds of each trial between groups, we assessed whether the lower accuracy in the LBP group in conditions with a large target was associated with a larger postural drift. LBP patients indeed tended to drift away from the target center more than healthy control subjects. Although this main effect of group did not reach significance in a repeated measures ANOVA including all experimental conditions ($p=0.077$), LBP patients showed on average a 37% (0.20^{o}) larger drift compared to healthy controls ($p=0.044$) in the condition with a large target and without lumbar muscle vibration (Figure 4.5).

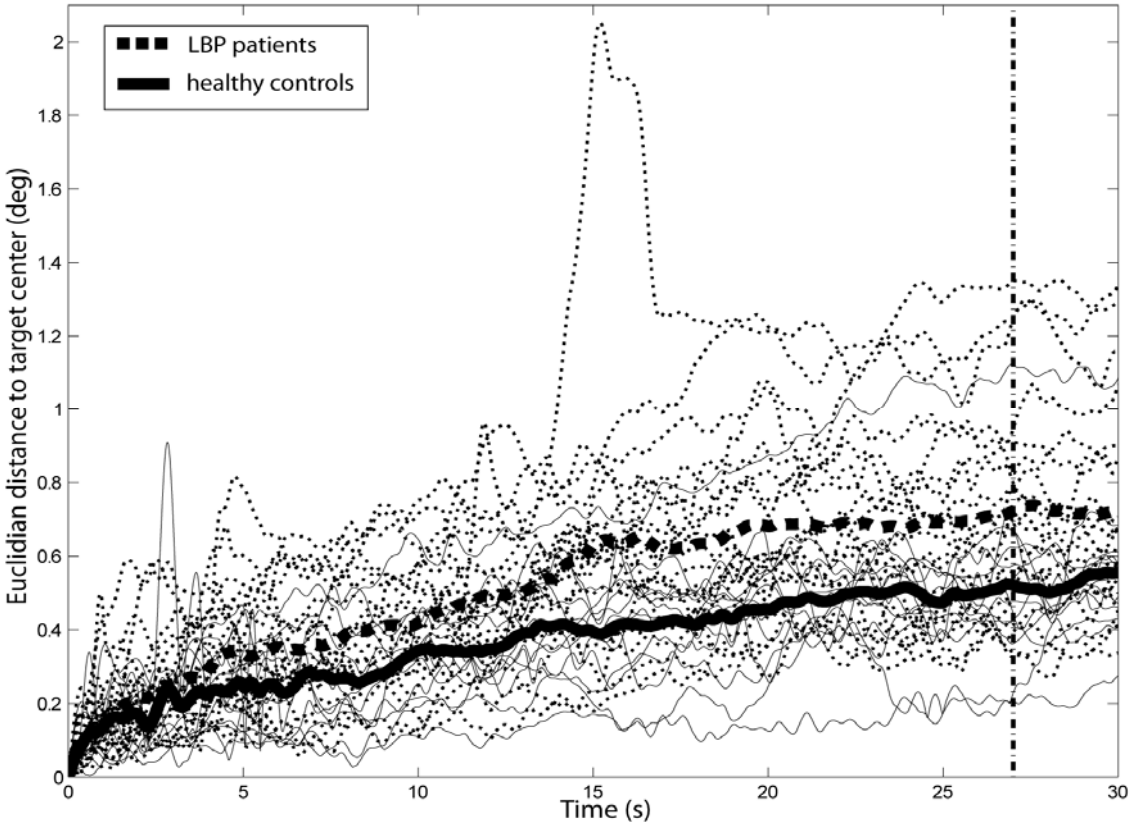

Figure 4.5. Time series of Euclidian distance to target center for LBP patients (dotted) and healthy controls (solid), averaged over two repetitions of the same condition with a large target and without vibration. Zero reflects each subject's self-chosen upright trunk angle at the start of the trial (the center of the target) and bold lines indicate group averages. The average distance over the last 3 seconds (indicated by the vertical dash-dotted line) quantified the postural drift with respect to the target center.

Figure 4.6 shows the co-activation ratio for both targets, vibration conditions, and groups. No significant main effect of group (p=0.830) or target (p=0.339) was found. Neither did we find the expected interaction between target size and group (p=0.920). A non-significant tendency towards an effect of vibration on co-activation ratio (p=0.089) suggested slightly higher co-activation ratios in conditions with lumbar muscle vibration. No significant interactions (all p≥0.293) were found.

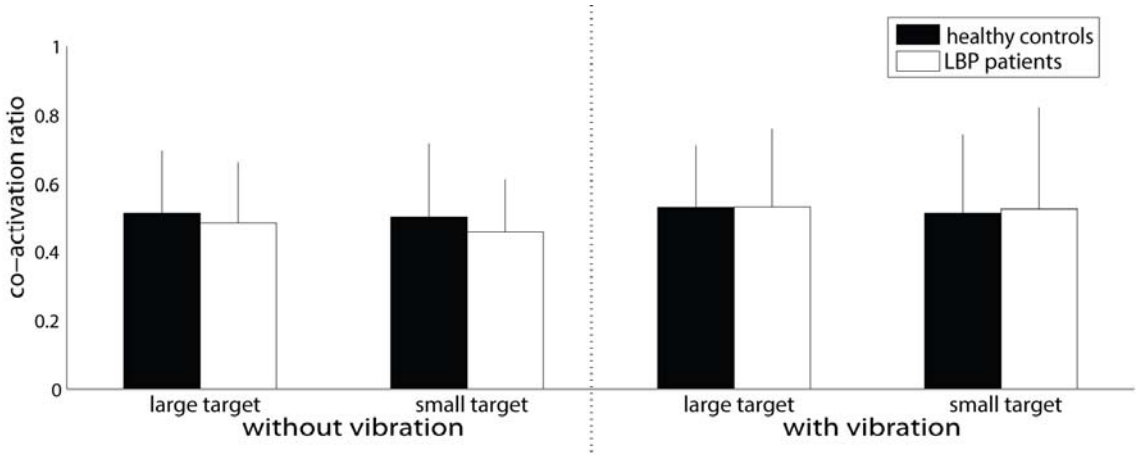

Figure 4.6. Ratios of co-activation (abdominal/back muscle EMG amplitudes) with error bars representing standard deviations over subjects within groups.

Discussion

We evaluated the control of an upright trunk posture in LBP patients and healthy control subjects and compared the effects of precision demands and lumbar muscle vibration on accuracy, precision, and trunk muscle co-activation between groups. When the precision demand was high, and visual feedback was available intermittently, LBP patients were able to control their trunk as accurately and precisely as subjects without LBP. However, when the precision demand was low and limited visual feedback was available, LBP patients were less accurate and tended to drift further away from the target center when compared to healthy subjects. In these same conditions, no difference between groups was found for precision. Effects of lumbar muscle vibration were similar between groups and no significant differences in trunk muscle co-activation were found between groups or conditions.

Interestingly, both LBP patients and healthy subjects with no (history of) LBP were able to reduce the standard deviation of trunk angle to about 0.1^{o} when the precision demand was high. However, a difference between groups was found in accuracy when the

precision demand was low. LBP patients showed a larger mean Euclidian distance to the target center, which tended to coincide with a larger postural drift. Since visual feedback of trunk angle was very limited when the target was large, this suggests that LBP patients were less able to detect low frequency drift by proprioception.

However, the deterioration of accuracy and precision when vibration was applied to the paraspinal muscles indicates that, in the control group as well as in LBP patients, proprioceptive input from lumbar muscle spindles was an important source of information for controlling trunk posture. This contrasts with previous findings, which suggest that LBP patients tend to rely less on proprioceptive feedback from lumbar muscle spindles [52, 54, 100]. These conflicting findings might be explained by the availability of alternative sources of feedback. In the present study, visual feedback of trunk angle was only provided when subjects left the target. This occurred very rarely in the condition with a low precision demand and only intermittently in the condition with a high precision demand. Moreover, trunk angle was defined by the position of an opto-electronic marker on the spinous process of T12 while the pelvis was fixated, so only movements in the lumbar spine contributed to trunk angle changes. Therefore, lumbar proprioception was probably the main source of relevant information, at least during a substantial part of the measurements. In contrast, in a previous study [100] we provided continuous visual feedback of trunk angle during a tracking task that required precise trunk movements, which allowed for reduced weighting of lumbar proprioception in LBP patients (be it at the cost of reduced precision). In the studies by Brumagne et al. (2004) and Claeys et al. (2011), vision was always occluded, but alternative sources of feedback were still available (e.g. vestibular information, pressure on support surface, proprioception from leg and upper trunk muscles), and postural sway was quantified by whole body center of pressure displacements [52, 54]. It seems that LBP patients only tend to rely on lumbar proprioceptive feedback as much as healthy subjects when alternative sources of relevant feedback are lacking.

The absence of any difference between groups in co-activation ratios indicates that LBP patients did not tend to stiffen their trunk more than healthy control subjects. One could argue that a ratio of co-activation (antagonist divided by agonist EMG amplitude) could disguise increased co-activation when amplitudes in both muscle groups are equally higher in LBP patients. Re-analysis of EMG amplitudes for abdominal and back muscle groups separately, however, did not reveal such increased EMG amplitudes in LBP patients.

Although previous studies have reported increased levels of trunk muscle co-activation in LBP patients [37-38], possibly reflecting a guarding strategy to protect the affected area, we did not find indications for the use of such a strategy in the current study. The same group of LBP patients did not show increased levels of antagonistic co-activation during a spiral tracking task either [100]. Apparently, both the current trunk positioning task and the previously reported spiral tracking task did not trigger protective muscle recruitment strategies. This may be due to the highly self-controlled nature of these precision tasks combined with the absence of external perturbations.

The finding that higher precision demands did not coincide with increased antagonistic co-activation is in contrast with previous findings on precision control in the limbs [43-45, 48], but supports previous findings on precision control of the trunk in healthy subjects [91] and low back pain patients [100]. Possibly, alternatives for trunk stiffening were preferred, due to the negative side effects of antagonistic co-activation (e.g. high metabolic costs, mechanical loading of the spine and interference with breathing). Another explanation could be that the net effect on precision of increased force variability of individual muscles on the one hand, versus increased joint stiffness associated with antagonistic co-activation on the other hand, which was positive in the elbow [42], may be negative in the trunk.

A closer look at the non-significant trend towards an effect of lumbar muscle vibration on co-activation ratio revealed that EMG amplitude averaged over the abdominal muscles tended to increase, while EMG amplitude averaged over the back muscles remained similar when vibration was applied to the paraspinal muscles. Rather than increased trunk stiffening by antagonistic co-activation, this increased abdominal muscle activation probably reflects a change in trunk posture. Specifically, vibration resulted in an offset towards extension (Figure 4) and thereby increased the gravity induced lumbar moment, which should be compensated by increased abdominal muscle activation. Importantly, these effects were similar between groups, so again no indications for changes in trunk muscle recruitment in the LBP patients were found.

A potential limitation of the present study is that we compared LBP patients and healthy controls at a group level, while inconsistent findings between previous studies suggest that not all LBP patients show motor behavior that is different from healthy subjects [22, 33]. However, scatter plots of all reported outcome measures did not reveal any

subgrouping of patients, which legitimized our group level approach. A second limitation is that while the experimental task in the current study allows for focusing on the area of interest and eliminates potential compensation mechanisms, it does not represent a functional task. Therefore, one should be careful in extrapolating the present findings to daily life. Nevertheless, the present findings add to our understanding of LBP, in that the weighting of proprioceptive feedback from lumbar muscle spindles in LBP patients seems to depend on the availability of other sources of relevant information.

To summarize, LBP patients were as accurate and precise as healthy control subjects in controlling a self-chosen upright trunk posture only when the precision demand was high and visual feedback was intermittently available. Paraspinal muscle vibration deteriorated trunk control both in the LBP patients and in the healthy control subjects, which suggests that the weighting of proprioceptive feedback from lumbar muscle spindles did not differ between groups. We therefore conclude that the difference between groups in accuracy on the large target is likely to point at a reduced sensitivity to postural drift in LBP patients.

Acknowledgements

The authors wish to thank Janneke de Bruin, Kim van Vijven, Nathalie Oomen and Sabrina Joosten for their assistance in subject recruitment and data collection.